Epidemiologic Study of Childhood Leukemia in Memphis and Shelby County, 1939–62

KATHLEEN HERNANDEZ, M.D., and GEORGE TOKUHATA, Ph.D., Dr.P.H.

AN INCREASE in leukemia death rates in recent decades has been reported by several investigators (*1–5*). From their reports and other related studies (*6–11*) certain epidemiologic characteristics of the disease have been identified. For example, leukemia is a more frequent cause of death among men than among women; it is primarily a disease of the extremes of life; white persons have a greater risk of dying from it than do Negroes; the disease is more prevalent among families of higher income; and it tends to aggregate in time and place of residence. The number of such studies, however, is not sufficiently large, and the results are not consistent enough to draw specific conclusions regarding the etiology of human leukemia. This circumstance indicates the need for more detailed studies of this disease.

The purpose of our study was to determine the incidence of childhood leukemia in Memphis and Shelby County, Tenn., for the 1939–62 period by race, sex, age, and family economic status. Data concerning the month of diagnosis and the place of residence will be presented in a separate paper. The total population under 20 years of age of Memphis and Shelby County was 87,590 in 1940; 123,663 in 1950; 224,014 in 1960. On the average, the study population included approximately 65 percent whites and 35 percent nonwhites (almost all Negroes). This population provided a favorable opportunity to study racial differences in the incidence of leukemia within a given community.

Dr. Hernandez was a postdoctoral epidemiology trainee, supported by National Cancer Institute training grant 1–T1–CA5176, at the time of the study. She is now instructor in pediatrics, St. Jude Children's Research Hospital, Memphis, Tenn. Dr. Tokuhata is chief of epidemiology at the hospital and assistant professor of preventive medicine, University of Tennessee, Memphis. *The study was supported by the American-Lebanese-Syrian Associated Charities.*

Materials and Methods

We examined the medical charts of all children under 20 years of age who were residents of Memphis or Shelby County, Tenn., during the years 1939 through 1962 and who were reported to have had leukemia during the period. Cases of lymphosarcoma were included in the study because, in children, lymphosarcoma often occurs as a phase of acute lymphocytic leukemia. The study patients had been admitted to one of the following hospitals in Memphis: John Gaston, Le Bonheur, St. Joseph, Methodist, Baptist Memorial, Crump, or Memphis Eye, Ear, Nose, and Throat. This listing includes all medical institutions in the community in which cancer diagnosis could be made during the period being studied. Death certificates at

the Memphis and Shelby County Health Department and medical records at the West Tennessee Tumor Registry were also used to complete the ascertainment of all reported cases of cancer. Information regarding race, sex, age, and place of residence by census tract, date of diagnosis, and leukemic cell type was abstracted from the hospital records. For the final analyses only cases for which a definite diagnosis was given were used.

The number of leukemia cases diagnosed annually in Memphis and Shelby County was not large enough to permit analysis by single years. Thus, for temporal comparison, the 24-year period was divided into three 8-year intervals, 1939–46, 1947–54, and 1955–62; cases of leukemia were grouped accordingly. The child population for Memphis and Shelby County, which was used as the denominator to compute the incidence rate, was taken from the 1940, 1950, and 1960 U.S. census reports. The rates were analyzed with respect to race, sex, and age and compared for the 8-year intervals.

To analyze the incidence of leukemia by economic status of the patient's family, the census tracts of Memphis and of the suburban residential area of Whitehaven were divided into upper and lower economic halves. The analysis by census tract was for the total population. The upper economic half, however, is assumed to have been almost entirely white and the lower, predominantly nonwhite. Data published in the 1940, 1950, and 1960 census reports on median property values and median contract monthly rents were used to determine economic status. Leukemia cases in the county areas were excluded from this analysis.

Results

During the 1939–62 period, 133 cases of childhood leukemia, including lymphosarcoma, were diagnosed, affecting 83 boys and 50 girls, or 97 whites and 36 nonwhites. All cases used in the final analyses were diagnosed as acute. The lymphocytic type of leukemia, including lymphosarcoma, represented by far the largest group, accounting for 58 percent of all cases (table 1). The proportion of the stem cell type was 23 percent and of the myelomonocytic type, 19 percent. There were no significant differences in the percentage distribution of cell types between the two sexes or between whites and nonwhites.

Incidence by race and sex. The incidence rates for leukemia per 100,000 of the population under 20 years of age by race and sex for each 8-year interval are given in table 2. For all races and sexes together, the rate rose sharply from 13.4 per 100,000 during the 1939–46 period to 36.9 during the 1947–54 period. This 2.8-fold increase is statistically significant. The rate declined to 27.4 during the 1955–62 period, a decrease not statistically significant. The mean annual incidence was 1.7 during the first period, 4.6 during the second, and 3.4 during the third.

In both boys and girls, the incidence of leukemia showed a sharp increase in the 1947–54 period. The trend was more marked, however, in boys (in whom the rate increased 3.2-fold) than in girls (whose rate increased 2.4-fold). In both sexes the increase was statistically significant. Both boys and girls experienced a slight decrease in incidence in the period 1955–62, but the change was not statistically significant in either sex.

Both white and nonwhite children experienced an increase in the risk of leukemia over the 24-year period. Outstanding race-sex differences in risk during the 1947–54 period were observed. Among white children, the increase in risk was much greater in boys (fourfold) than in girls (twofold); among nonwhite children, however, the increase in risk was much greater in girls (fivefold) than in boys (twofold).

The sex ratio was computed as the incidence rate for boys to the incidence rate for girls; the race ratio, as the incidence rate for whites to the incidence rate for nonwhites (table 2). The sex difference increased consistently over the 24-year period. During the first 8-year period the sex ratio was 1.19; there was only a slight predominance of boys with leukemia over girls with the disease. The sex ratio then rose to 1.58 during the second period, and finally to 1.91 during the third. By the third period childhood leukemia had become almost twice as frequent in the boys as in the girls.

A remarkable difference was noted in the direction of the change in incidence of leukemia between white and nonwhite children. During

the 1939–46 period, more white girls than white boys were affected by the disease; in contrast, the disease was much more prevalent among nonwhite boys than among nonwhite girls. During the succeeding two 8-year periods, the sex difference consistently increased among the white children but consistently decreased among the nonwhite. Thus, leukemia became predominant in boys within the white group, whereas the sex difference disappeared within the nonwhite group.

On the other hand, leukemia was more prevalent among white than among nonwhite children in all three periods, and this difference gradually increased in the more recent study years. The race ratios for the three periods were 1.68, 1.77, and 2.05; by the third period, the risk of white children developing leukemia had become at least twice as great as that of nonwhite children. An interesting difference in the direction of the change among boys and girls was observed. Specifically, the risk of developing leukemia increased faster for white boys than for nonwhite boys, and by the third period had become at least three times greater for white boys. In contrast, this risk increased faster for

Table 1. Cases of leukemia and percentage distribution, by cell type, race, and sex, Memphis and Shelby County, Tenn., 1939–62

Cell type	Total		Male		Female		White		Nonwhite	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
All types	133	100.0	83	100.0	50	100.0	97	100.0	36	100.0
Lymphocytic (including sarcoma)	77	57.9	49	59.0	28	56.0	55	56.7	22	61.1
Stem cell	31	23.3	18	21.7	13	26.0	23	23.7	8	22.2
Myelomonocytic	25	18.8	16	19.3	9	18.0	19	19.6	6	16.7

Table 2. Incidence of leukemia per 100,000 persons under 20 years of age, by race and sex, Memphis and Shelby County, Tenn., 1939–46, 1947–54, and 1955–62

Race and sex	1939–46 (*X*)		1947–54 (*Y*)		1955–62 (*Z*)		*X*−*Y*		*Y*−*Z* change in rate
	Rate	Cases	Rate	Cases	Rate	Cases	Change in rate	*P*	
All races	13.4	15	36.9	46	27.4	72	2.75	0.0002	0.74
Male	14.6	8	46.1	28	35.8	47	3.16	.0020	.78
Female	12.3	7	29.0	18	18.8	25	2.36	.0466	.65
White	16.5	10	44.6	33	34.8	54	2.70	.0040	.78
Male	13.2	4	55.1	20	49.6	39	4.17	.0044	.90
Female	19.7	6	34.3	13	18.9	15	1.74	.2542	.55
Nonwhite	9.8	5	25.3	13	16.9	18	2.58	.0588	.67
Male	16.3	4	32.2	8	15.2	8	1.98	.2542	.47
Female	3.8	1	18.2	5	18.6	10	4.78	.1164	1.02
Sex ratio:[1]									
All races	1.19		1.58		1.91				
White	.67		1.60		2.63				
Nonwhite	4.28		1.76		.81				
Race ratio:[2]									
Both sexes	1.68		1.77		2.05				
Male	.80		1.71		3.26				
Female	5.18		1.89		1.01				

[1] Male incidence rate to female incidence rate.
[2] White incidence rate to nonwhite incidence rate.

NOTE: U.S. census populations of 1940, 1950, and 1960 were used as denominators in computing rates.

nonwhite girls than for white girls. During the 1955–62 period the risk for the two groups of girls became equal.

Incidence by age group. The incidence of leukemia was computed in four age groups: children under 5 years, 5–9 years, 10–14 years, and 15–19 years. The age-specific rates for the intervals 1939–46, 1947–54, and 1955–62 are summarized in table 3.

As expected, throughout the 24-year period the highest rate of leukemia was found in the youngest group. Gradually lower rates were recorded for older children. During the 1947–54 period, the rate per 100,000 was 48 among children under 5 years of age, 38 in the 5–9 year age group, 29 in the 10–14 year age group, and 24 in the 15–19 year age group. The 8-year rates correspond to the following mean annual rates during the same period: 6.0, 4.8, 3.6, and 3.0.

In all age groups considered, the incidence of leukemia rose remarkably during the second period, but became generally stable during the third. There were considerable variations in the relative change that was observed during the second period. The increase in the 10–14 year group was as large as 4.2-fold, whereas the increase in the youngest group was only 1.8-fold. The increase was 3.7-fold in the 15–19 year group and 2.5-fold in the 5–9 year group. The largest increase observed, in the 10–14 year group, was statistically significant, but the increases observed in the 15–19 and 5–9 year groups were of borderline significance. The overall rise in leukemia incidence during the 24-year period was 3.2-fold for the 15–19 year group, 1.7-fold for the group under 5 years and for the 5–9 year group, and 1.5-fold for the 10–14 year group.

A more detailed analysis was performed within each of the four age groups for the same three 8-year periods with respect to race and sex (table 4).

First, we analyzed the relative change in the incidence rate. Since the major change occurred between the 1939–46 and the 1947–54 periods, our detailed analysis was limited to these periods. Among all children under 5 years of age, the 1.8-fold increase in incidence during these periods was largely accounted for by nonwhite children, who experienced a 3.6-fold increase, and by boys, who experienced a 2.6-fold increase (table 4). In the 5–9 year group, the rise was largely attributable to boys, whose risk of developing leukemia increased by as much as 5.8-fold. In the 10–14 year group, a larger increase was recorded among white than nonwhite children and among girls than boys. Similar race and sex differences were noted in the 15–19 year group. Two generalizations can be made. During early childhood, particularly in children under 10 years of age, the incidence of leukemia increased faster among nonwhites than whites and among boys than girls. During late childhood, particularly from ages 10 through 19 years, leukemia increased faster among whites than nonwhites and among girls than boys.

Second, we analyzed the sex ratio and the race ratio in each of the four age groups. These ratios were based on the incidence rates computed per 100,000 of sex-specific or race-specific child populations. In general, the race difference tended to decline as the children's age

Table 3. Incidence of leukemia per 100,000 persons under 20 years of age by age groups, Memphis and Shelby County, Tenn., 1939–46, 1947–54, and 1955–62

Age group (years)	1939–46 (X)		1947–54 (Y)		1955–62 (Z)		$X-Y$		$Y-Z$ change in rate	$X-Z$ change in rate
	Rate	Cases	Rate	Cases	Rate	Cases	Change in rate	P		
Under 5	26. 7	7	47. 8	21	45. 4	36	1. 79	0. 1770	0. 95	1. 70
5–9	15. 4	4	38. 4	12	26. 5	19	2. 49	. 0990	. 69	1. 72
10–14	6. 9	2	29. 0	7	10. 0	6	4. 20	. 0500	. 34	1. 45
15–19	6. 5	2	23. 8	6	20. 8	11	3. 66	. 0872	. 87	3. 20

NOTE: U.S. census populations of 1940, 1950, and 1960 were used as denominators in computing rates.

advanced: during early childhood white children had a greater risk of developing leukemia than did nonwhite children. This difference became somewhat less marked in the period 1959–62. In children under 5 years of age, the predominance of white children over nonwhite in incidence of leukemia was as large as fivefold during the 1939–46 period. This difference declined, however, to approximately twofold during the following two periods. In children over 10 years of age, the racial difference in incidence was less noticeable and, inconsistently, the difference was reversed in the 15–19 year age group, in which nonwhite children had a greater risk.

Although during the 1939–46 period the overall incidence of leukemia did not differ between the two sexes, there was a clear sex difference according to age group (table 4). Under 10 years of age, more girls than boys were affected by leukemia, whereas from 10 through 19 years more boys than girls were affected. During the 1947–54 period, the sex difference increased, and more boys than girls were affected in all age groups. This trend generally remained during the 1955–62 period.

Two features regarding the sex difference may be emphasized. Within the 5–9 year group, the sex difference increased consistently and markedly during the entire 24 years, from 0.34 in the first period, to 1.41 in the second, to 5.27 in the third. These data mean that in the 5–9 year group the risk for boys of developing leukemia became at least five times greater than that for girls. Within the 15–19 year group, however, the sex difference changed consistently in the opposite direction, from at least 13.8 in the first period, to 1.27 during the second, to 0.91 in the third. Thus, in the 15–19 year group there was no longer an appreciable sex difference in the risk of leukemia.

Incidence by economic status. We analyzed the incidence of leukemia according to the economic status of the patient's family; the age factor was also taken into consideration. All children in the upper economic group had in general a greater risk of developing the disease than children in the lower (table 5). This difference increased gradually, becoming most conspicuous in the 1955–62 period, during which 36 children per 100,000 in the upper economic group, as compared with 19 per 100,000 in the

Table 4. Incidence of leukemia per 100,000 persons under 20 years of age by race and sex in different age groups, Memphis and Shelby County, Tenn., 1939–46, 1947–54, and 1955–62

Age group (years), race, and sex	1939–46 (X)		1947–54 (Y)		1955–62 (Z)		Change in rate	
	Rate	Cases	Rate	Cases	Rate	Cases	$X-Y$	$Y-Z$
Under 5:								
White	43.6	6	60.0	16	58.7	26	1.38	0.98
Nonwhite	8.1	1	28.9	5	28.7	10	3.57	.99
Male	22.7	3	59.0	13	55.2	22	2.60	.94
Female	31.1	4	36.5	8	35.6	14	1.17	.98
5–9:								
White	21.8	3	48.6	9	39.1	16	2.23	.80
Nonwhite	8.2	1	23.5	3	9.7	3	2.87	.41
Male	7.8	1	44.9	7	44.3	16	5.76	.99
Female	22.8	3	31.9	5	8.4	3	1.40	.26
10–14:								
White	0	0	29.8	4	14.0	5	0	.47
Nonwhite	15.3	2	28.0	3	4.2	1	1.83	.15
Male	13.9	2	41.9	5	13.4	4	3.01	.32
Female	0	0	16.4	2	6.7	2	0	.41
15–19:								
White	5.7	1	26.7	4	19.1	7	4.68	.72
Nonwhite	7.6	1	19.5	2	23.9	4	2.57	1.23
Male	13.8	2	27.0	3	19.8	5	1.96	.73
Female	0	0	21.2	3	21.8	6	0	1.03

NOTE: U.S. census populations of 1940, 1950, and 1960 were used as denominators in computing rates.

Table 5. Incidence of leukemia per 100,000 persons under 20 years of age by economic status of the family and by age group, Memphis, Tenn., 1939–46, 1947–54, and 1955–62

Economic and age group (years)	1939–46 (X)		1947–54 (Y)		1955–62 (Z)		Change in rate	
	Rate	Cases	Rate	Cases	Rate	Cases	$X-Y$	$Y-Z$
Upper economic half	14.3	5	40.2	20	36.2	39	2.81	0.90
Under 5	38.6	3	54.7	10	59.1	19	1.42	1.08
5–9	0	0	48.6	6	36.9	11	--------	.76
10–14	11.0	1	22.7	2	19.7	5	2.06	.87
15–19	9.5	1	19.8	2	20.5	4	2.08	1.04
Lower economic half	15.9	8	27.9	21	18.6	22	1.75	.67
Under 5	24.9	3	27.7	7	33.8	13	1.11	1.22
5–9	42.0	5	31.1	6	17.3	6	.74	.56
10–14	0	0	26.2	4	0	0	--------	--------
15–19	0	0	26.5	4	15.7	3	--------	.59

NOTE: U.S. census populations of 1940, 1950, and 1960 were used as denominators in computing rates. Significance tests between the economic halves (total group under 20 years of age) were as follows: 1939–46, $P>0.05$; 1947–54, $P>0.05$; and 1955–62, $P=0.0086$. The significance tests between 8-year periods (total group under 20 years of age) were as follows: $X-Y$ (upper economic half), $P=0.0292$; $Y-Z$ (upper half), $P>0.05$; $X-Y$ (lower economic half), $P>0.05$; and $Y-Z$ (lower half), $P>0.05$.

lower, developed leukemia. This difference was statistically significant. No particular economic difference was noted during the 1939–46 period.

In general, the incidence of childhood leukemia changed in the same direction over time in both upper and lower economic groups. During the earlier study years, however, children of the upper economic group experienced a greater increase (2.8-fold) than children of the lower economic group (1.8-fold). During the more recent study years the rate declined slightly in both groups, but none of these changes was statistically significant.

The incidence of leukemia among children under 5 years of age increased steadily in both economic groups throughout the entire 24-year period (table 5). Further, the increase was greater for the upper economic group during the earlier years, whereas the increase was greater for the lower during the more recent years.

In the lower economic group, the risk of developing leukemia during the 1939–46 and 1947–54 periods was greater among children between 5 and 9 years than among those in the youngest age group. Whether this unusual difference reflects the rather small number of cases reported during the earlier study years is not clear. This difference gradually diminished thereafter, however, and was finally reversed during the 1955–62 period. Children in the 5–9 year age group in lower economic families continuously experienced a declining incidence of the disease throughout the entire 24-year period, whereas children in upper economic families did not.

Discussion

Memphis has long had a large and comprehensive medical center consisting of the University of Tennessee College of Medicine and a number of public and private hospitals, including pediatric institutions. It is unlikely that in a significant number of cancer cases the diagnosis was missed, except possibly during the early years of the study period. There was no evident difference between white and nonwhite children in regard to accessibility to pediatric services in the Memphis area. While the 1950 census report represents a population at midpoint for the 1947–54 period, the 1940 and 1960 reports were used as the best available population estimates for the 1939–46 and 1955–62 periods. This method of computation might have affected the interperiod comparison of the rates. To check on these possible influences we therefore reanalyzed the same data for the three 3-year intervals 1939–41, 1949–51, and 1959–61, using the 1940, 1950, and 1960 census populations as denominators. The results were not different,

however, from those of the earlier analysis in which 8-year intervals were considered.

The most notable observations in this study were that the incidence of childhood leukemia increased remarkably after World War II, particularly beginning in 1948, and that the rate stabilized in the later study years. Children with different characteristics with respect to race, sex, age, and economic status had different experiences with respect to leukemia. While boys had a greater risk of developing the disease than girls, boys also experienced a larger relative increase in the incidence rate.

The risk for white children of developing leukemia was consistently greater than that for nonwhite children. Further, among white children the rate increased faster in boys, whereas among nonwhite children the rate increased faster in girls.

Although the highest risk was found in the youngest age group, a larger relative increase was observed in older age groups, particularly in the 10–14 year group. In children under 10 years, the leukemia rate increased faster among nonwhites as a whole and among boys as a whole. In contrast, in children from 10 through 19 years, the rate increased faster among whites as a whole and among girls as a whole.

During the 1939–46 period no differences were observed in the incidence of leukemia between the lower and upper economic halves. In the years following, however, children of the upper economic class showed an increasingly greater risk of the disease.

Undoubtedly, multiple factors have contributed to the recent rise in the incidence of childhood leukemia, including improvements in case-finding and increased exposure of children to causative agents. It is difficult to determine to what extent improved diagnostic techniques and better availability of medical care have been responsible. The amount and pattern of increase observed in different groups suggest, however, that the true incidence of this disease in the population under study did rise considerably.

The increase in the true incidence of leukemia can be attributed to both host and environmental factors, some of which will be discussed briefly.

1. Introduction of antibiotics and sulphonamides against infectious disease, as well as improvement in nutrition and general living conditions, has resulted in a prolonged survival of children. Children of certain ages surviving infectious diseases in the current decade may have had different genetic-constitutional make-ups than children of the same ages surviving similar diseases in an earlier decade. Those who survive infectious diseases are subject to the risk of developing chronic diseases, particularly leukemia, during early childhood. The usual method of age-adjustment cannot be used to answer this question of selective survival in relation to leukemia risk. A high inverse correlation between leukemia deaths and pneumonia deaths in a child population was clearly demonstrated in England for the years following World War II (*12*). A similar correlation can undoubtedly be demonstrated in the population under study.

2. The use of X-rays and radioisotopes for diagnosis and therapy has increased in recent years. The leukemogenic effect of ionizing radiation has been demonstrated in animals and implicated in human beings. Evidence in respect to man has been provided by a number of studies—of the atomic bomb survivors, of children with a history of prenatal X-ray exposure, of patients with ankylosing spondylitis, hyperthyroidism, or enlarged thymus who have been treated with radiation, and by studies of longevity and mortality among radiologists (*13–18*). While the leukemogenic effects of ionizing radiation in man are not yet completely understood, only a relatively small proportion of childhood leukemia cases may be accounted for by such an agent.

3. The use of toxic chemicals such as insecticides, household chemicals, paint, hair sprays, tobacco smoke, and a variety of drugs which are potentially toxic has greatly increased in recent years. These chemicals may be directly or indirectly leukemogenic, particularly in children. A significant association between benzene and leukemia has been reported (*19*). Carcinogenicity of tobacco smoke in animals has been documented (*20*), and the same effect may hold true for human beings. The proportion of smokers in the population has steadily increased, and since this trend has been more marked among young women, the number of fetuses, infants, and young children exposed to tobacco

smoke, either directly or indirectly, has also increased. The importance of such exposure in childhood leukemia, however, is yet to be determined.

4. A marked increase in the number of industries in Memphis and the vicinity has created industrial pollution of the air and water in some sections of the city, and in certain of these sections a higher than average rate of leukemia has been observed among residents.

5. As the population of Memphis has grown in recent decades, residential crowding has also increased. If a virus or any other transmissible agent is involved in leukemogenesis in man, an association between the rise in the incidence of leukemia and the increase in the frequency of personal contacts might be expected. In other studies (*21*, *22*), the incidence of leukemia and population density were found to correlate. Further, the number of families possessing dogs has risen markedly during the past two decades in both Memphis and Shelby County, and these household animals could also serve as vectors in transmitting leukemogenic agents to children.

Summary

The incidence of childhood leukemia in Memphis and Shelby County, Tenn., for the 1939–62 period was analyzed with respect to race, sex, age, and family economic status. A total of 133 cases of leukemia in persons under 20 years (83 boys and 50 girls, 97 white and 36 nonwhite) were used in the final analysis. Cell types did not differ between the two sexes or between the two racial groups. The incidence of the disease increased markedly after World War II, but the rate has stabilized in more recent years.

While boys had a greater risk of developing leukemia than girls throughout the entire study period, boys also experienced a larger relative increase after World War II. The rate was consistently higher among white children than among nonwhite children. Among white children, the rate increased faster in boys; among nonwhite children, it increased faster in girls. Although the greatest risk was found in the youngest age group, a larger relative increase was observed in the older age groups.

During the earlier study years, the leukemia incidence did not differ between the upper and the lower economic groups. The rate became increasingly higher in the upper economic group, however, during the later study years. The upper economic group represented almost entirely white children, and the lower group, mostly Negro children.

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Public Health Service Staff Appointments

Dr. William L. Kissick has been appointed chief of the Division of Public Health Methods, Public Health Service. Dr. Kissick has been deputy chief of the Division.

Born in Detroit, Mich., Dr. Kissick received doctoral degrees in medicine and epidemiology from Yale University. He was commissioned in the Public Health Service following a residency in social medicine at Montefiore Hospital and the New York City Department of Health.

Dr. Kissick has served as executive secretary, Second National Conference on Public Health Training, 1962–63; deputy chief, Office of Research Grants, Bureau of State Services, 1963–64; and special assistant to the Assistant Secretary for Health and Scientific Affairs, Department of Health, Education, and Welfare. He has had consultant, advisory, and special staff appointments with the National Commission on Community Health Services; President's Commission on Heart Disease, Cancer, and Stroke; Office of Economic Opportunity; Appalachian Regional Commission; Institute for Policy Studies; and Georgetown University School of Medicine. He was a member of the Delegation on Medical Education that studied educational and research institutions in the Soviet Union.

Dr. Kissick is a diplomate of the American Board of Preventive Medicine and a member of numerous professional associations.

Dr. Heinz Specht has been appointed chief of the Office of International Research, National Institutes of Health, Public Health Service. Dr. Specht has been assistant chief for scientific affairs, Office of International Research, since August 1965. He was previously chief of the Institutes' Pacific Office in Tokyo, Japan.

A native of New York State, Dr. Specht is a graduate of Princeton University and holds a Ph.D. in physiology from Johns Hopkins University. He has been with the Institutes since 1936.

In his new position, Dr. Specht will be responsible for development of Institutes' policy in international activities and coordination of Institutes' programs which involve other countries. He will also be responsible for liaison of the Institutes with countries cooperating in biomedical research.

Dr. Specht is president-elect of the Washington Academy of Sciences.